# Pulmonary Circulation

## Introduction

This lesson explores the pulmonary vasculature with an emphasis on the arterial supply and capillaries. We'll start off with a comparison of systemic and pulmonary circulation to introduce the whys and whats of pulmonary circulation. Next, we'll lay out our approach to this material, focusing on how pushing and pulling are used to change the diameter of a blood vessel, thereby altering the resistance and flow, something that did not need to be considered in cardiology when studying the systemic vasculature. We'll then explore pulmonary vascular resistance, the effects of hypoxia on vascular resistance, the effects of gravity and the zones of the lungs on vascular resistance, and, finally, how exercise can used to demonstrate recruitment and distension of pulmonary vessels. This information is going to be useful for the lessons that follow regarding V/Q mismatch and pulmonary hypertension.

The beginning of this lesson is going to feel like a lot of, "*this is this, and that is that,*" seemingly useless factoids of ephemeral physiology that serve no purpose. The reason for that is, on their own, they are. But you need all the pieces up front in order to put them together in the end. You know OME by now. You know we aren't just going to regurgitate what others have done the way they've done it. The payoff is huge, but you do have to get through it.

We also hope that you'll find yourself saying, *"yeah, yeah—I know this already."* **Read the stuff you already know anyway.** What we've done is made pulmonary intuitive if you already know the other stuff. We only include the other stuff for you to draw a natural conclusion about the pulmonary stuff. So read everything, even if you already know it, so that you have exactly the pieces you need for the pulmonary conclusion.

## Systemic vs. Pulmonary Circulation

The pulmonary circulation is vastly different from the systemic circulation. This is because the blood doesn't have that far to travel (right heart to left heart), and there is a need to keep the pressures down in the lung. The equations that matter are different, the vessels are different, and the right ventricle that feeds the pulmonary artery is different from the left ventricle that feeds the aorta. We shall explain, but let's first review systemic circulation.

Blood fills the left atrium, mostly due to gravity. There is very little pressure in the pulmonary vein, but what little there is opens the mitral valve in diastole. Throughout systole, the aortic valve was open. Now, with it closed the large arteries that had distended recoil back on the lumen, driving blood forward (because the aortic valve prevents backflow). At the end of diastole, the pressure in the arteries is 80 mmHg. Blood fills the left ventricle during diastole. Systole starts. Blood is ejected into the systemic circulation and goes to every organ. The pressure at the peak of systole is high, 120 mmHg, to ensure adequate perfusion pressure. The mean arterial pressure is 90 mmHg. The large arteries feed the medium arteries, which in turn feed the small arteries. Across these arteries, the mean arterial pressure is kept relatively constant. The point is that these conducting arteries are moving blood from the heart to every organ, including the most distal ones. This is a great distance to cover, so the high pressure ensures the adequate delivery of blood. Each arteriole knows what their capillary bed needs in terms of pressure. The conducting arteries brought more than enough perfusion pressure. The arteriole's job is to crank up the resistance, thereby dropping the pressure from what was needed to ensure blood delivery down to what the filtration forces require. The point of the systemic circulation is to deliver enough pressure to promote **filtration**. The equation that matters in the systemic capillary bed is $\mathbf{J = k\,(H - O)}$, the forces of filtration. The goal is to push fluid and oxygen out into the tissue on the arteriole side of the

capillary and then to reabsorb fluid on the venule side. Oxygen does come off of hemoglobin and carbon dioxide loads onto hemoglobin (which, as we will discuss, is very much similar to the purpose of the pulmonary circulation). But the equation that matters most to the peripheral tissues is for filtration, not gas exchange. In order to resist such high pressures, conducting arteries are **large,** have a thick smooth muscle layer, **and, most importantly, have lots of elastin**. This enables them to distend in systole and recoil in diastole. In contrast, to crank up the resistance, arterioles have **a thick smooth muscle layer** relative to the size of their lumen. The smooth muscle and elastin are also necessary to ensure the adequate delivery of blood and regulation of hydrostatic force.

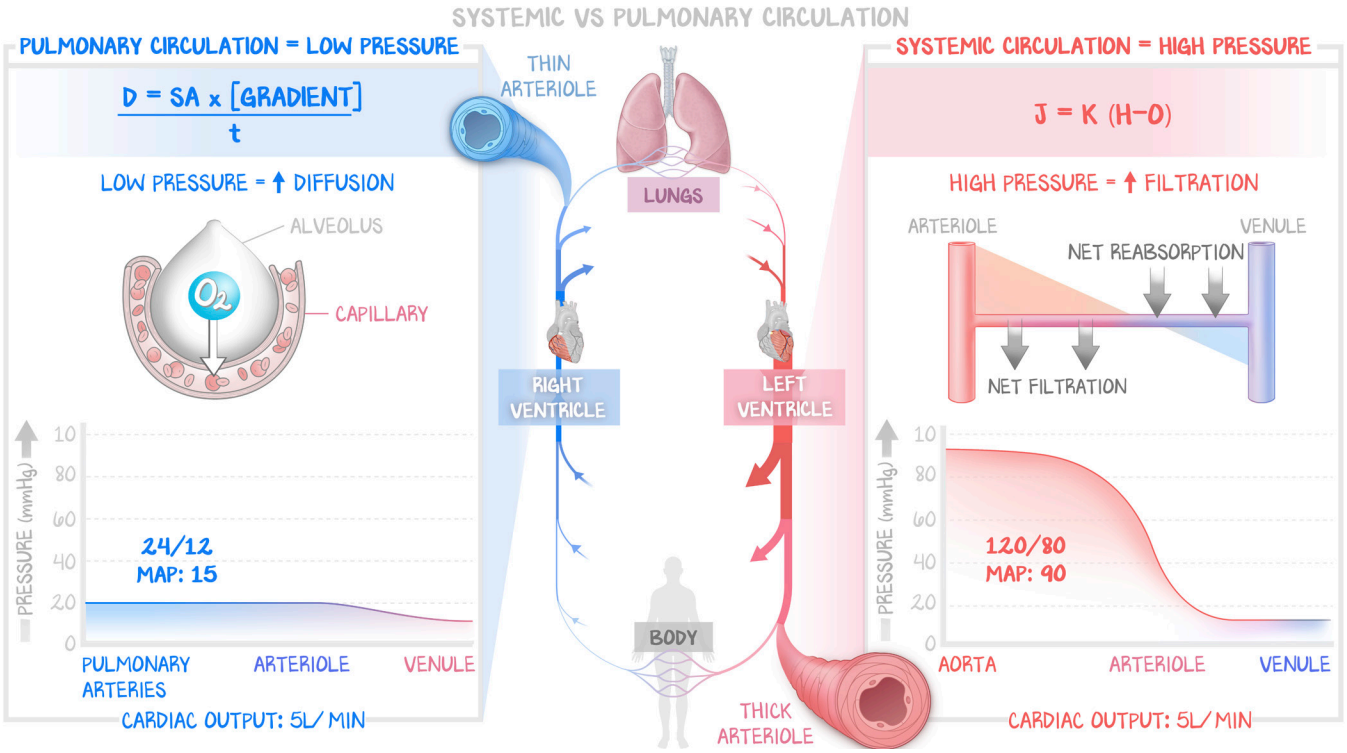


**Figure 1.1: Systemic vs. Pulmonary Circulation**
Systemic circulation is a high-pressure system. High pressure ensures adequate perfusion to all arterioles, even those furthest from the heart. Systemic arterioles are designed to crank up the resistance to drop that perfusion pressure to just what their capillaries need. In systemic tissues the goal is to use that remaining pressure to filter and reabsorb. The pulmonary circulation can make due with much less perfusion pressure to get blood to where it needs to go—the alveoli—which are already quite close to the right ventricle. The alveolar capillaries aren't built for filtration and reabsorption, but rather for the gas exchange. Thus the pressure and the equations we use to discuss them, are different, even though they are still arterioles leading to capillaries.

Now let's consider the pulmonary vasculature. There is only a **small distance** to travel from the right ventricle to the pulmonary capillaries. All of the blood from the right ventricle goes only to the pulmonary capillaries. Thus, there is no need to have a lot of pressure—the high systemic pressure is to ensure adequate delivery of blood to all organs. If there isn't a need for a lot of pressure, then the conducting arteries don't need a lot of smooth muscle to resist the force of the contraction of the heart. They do still have some elastin, to maintain the MAP during diastole, but they don't need a thick tunica media. If the conducting arteries don't deliver excess perfusion pressure to the arterioles, the arterioles don't need to crank up their resistance. They still want to get the capillaries' hydrostatic pressure down, so they still need smooth muscle, just not as much as those in the systemic vasculature. **This means the vessels take up less space.** And since the lung's purpose is to inflate to bring in fresh

oxygen, making more room for the alveoli to do that makes a lot of sense. The equation that matters in the alveolar capillary is the diffusion of gas equation, (D = SA × [gradient] / thickness), and not the filtration equation. The goal is to **minimize the thickness** of the diffusion barrier and **maximize the concentration gradient**. Filtration is bad at the alveolar capillary—the extravasation of fluid we call pulmonary edema. Thus, in order to prevent filtration and ensure a minimal oxygen diffusion barrier, the **pressure needs to be low**. The need for low pressure in the capillaries, the short distance to travel, and the guarantee of delivering the blood to the right capillaries means the **right heart doesn't need to generate a lot of pressure**. The normal pulmonary pressures are **24/12** with a **MAP of 15**. Cardiac output is **5 L/min**, and that is how much the right heart pumps. The same amount as the left ventricle, just not as hard, and so doesn't need as much muscle.

## Push- and Pull-forces from the Lung

The pulmonary vasculature is low pressure—24/12, MAP of 15. That means that small alterations in pressure make a difference. Think about what you know about the arterioles and venules. An increase in hydrostatic pressure because of right heart failure results in edema. Where do you get edema? The lower extremities. And it isn't anything about the lower extremities that causes the edema to go there. It is that they spend most of the day inferior to the rest of the body. Gravity causes dependent edema. Gravity causes venous pooling in the legs. The pressure in the veins can be negative (i.e., the flow is away from the heart) due to gravity. Now, gravity can make a difference to a diseased artery with a pretty bad blockage (as we saw in peripheral vascular disease), but it usually doesn't because nondiseased vessels have so much perfusion pressure that the arterioles' job is to generate a lot of resistance to bring it down to the pressures the capillaries can tolerate.

So, that means the pulmonary veins, like the systemic veins, can fall victim to small changes in pressure. Pressure. We've talked about pressure changes in the Lung series already. Let's review quiet respiration. The diaphragm contracts, the volume of the alveoli increases, so the pressure goes down, and air gets sucked in. The diaphragm contracts, so pulls on the pleura, decreasing the pleural pressure. The diaphragm contracts, indirectly pulling on alveoli, which are connected to other alveoli and to the bronchioles, and their volumes increase, too. Increasing the luminal radius of the bronchioles reduces the resistance. Less resistance, more flow. It is easy to get air into the lung. The diaphragm relaxes; the elastic recoil of the alveoli causes them to collapse, reducing the volume. As the volume is reduced, the pressure goes up, and air is pushed out of the alveoli. The force that pulled open the bronchioles is gone, and they collapse, too. Decreasing the lumen radius of the bronchioles increases resistance. More resistance, less flow. It isn't a problem for healthy lungs, but it was the defining pathophysiology of emphysema.

Now watch. You're satisfied with the effect of push and pull on the bronchioles, right? What's a bronchiole? It's a circular structure lined with epithelium through which a fluid (air) flows. What's an arteriole? Or a venule? They are circular structures lined with epithelium through which a fluid (blood) flows. The bronchioles are **extra-alveolar** ducts. The arterioles and venules are also **extra-alveolar** ducts.

**The pulling forces** of the lung **dilate the arterioles and venules**, increasing their radius, decreasing the resistance**, increasing blood flow**. The pulling force of the lung is inspiration; inspiration opens extra-alveolar vessels.

**The pushing forces** of the lung **compress the arterioles and venules**, decreasing their radius, increasing the resistance, **decreasing blood flow**. The pushing force of the lung is expiration; expiration closes extra-alveolar vessels.

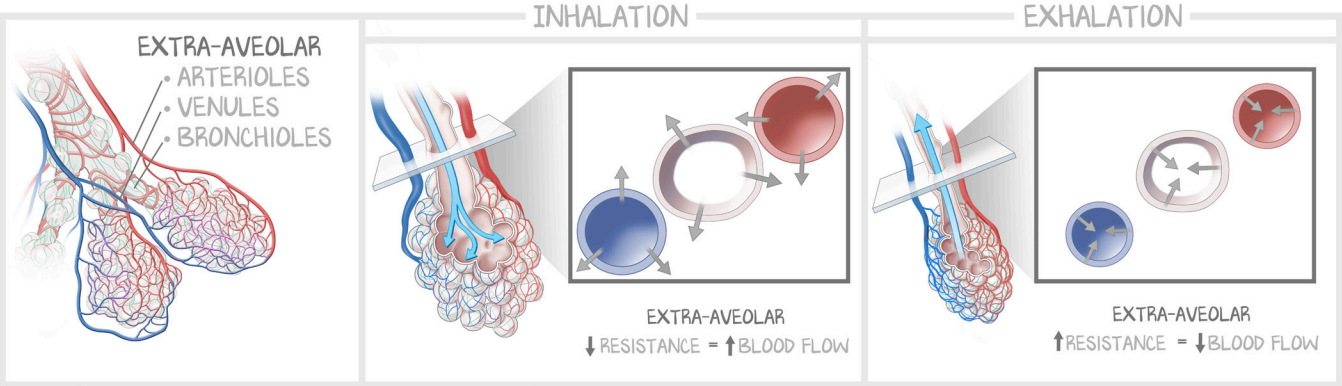


**Figure 1.2: Next, Add the Arterioles and Venules**
This figure shows something that should be familiar to you. You saw it in the lesson on the mechanisms and regulation of ventilation. Because the bronchioles have no cartilage, when the diaphragm contracts, they open. When the diaphragm relaxes, they collapse, but only a little. Force a lot of air out all at once, and they collapse a lot. Venules and arterioles, extra-alveolar vessels, behave like the bronchioles. They are pulled open on inspiration with less resistance and greater flow, and collapse on expiration with increased resistance and decreased flow.

And lastly, what about the **capillaries**? It's the **opposite** of the -ioles. When alveoli collapse, they push out their air. But as they collapse, they are **pulling on the septa**, pulling on the capillaries within the septa, opening them up, reducing resistance. When the alveoli fill with air, they **compress the septa,** pushing on the capillaries of the septa, closing them down, increasing resistance.

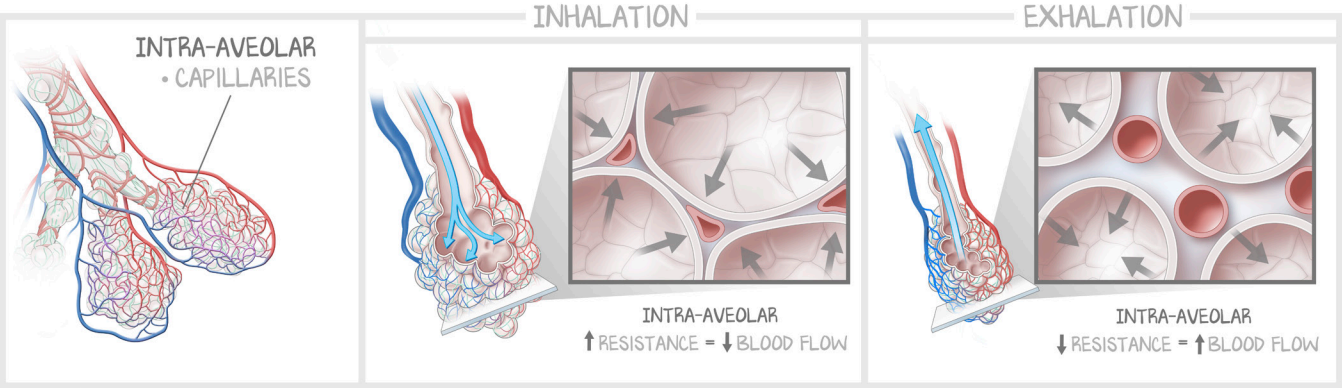


**Figure 1.3: Next, Add the Capillaries**
The intra-alveolar vessels, the capillaries, are compressed when the alveoli expand and pulled open when the alveoli collapse. That means they have the exact opposite relationship to that of the alveolar vessels. Resistance in the alveoli is highest when at maximum inhale (so flow is the lowest) and lowest when at maximum exhale (so flow is highest).

## Push-forces from within Vessels

Let's go back to Renal, to the glomerular capillary, the capillary of filtration. Renal blood flow (RBF) is the perfusion pressure that enters the glomerular capillary. Some of it is translated into glomerular filtration rate (GFR, enters the tubules), and some of it is translated into tubular blood flow (TBF, an OME term only, enters the tubule capillaries). If more RBF enters the system, and the resistance of the arterioles doesn't change, then there is going to be more GFR and more TBF. How did GFR go up? Through an **increase in the hydrostatic force** coming from inside the capillary, pushing water, ions, and other small molecules through leaky, fenestrated capillaries. **More pressure in pushed more fluid out.**

Come back to our pulmonary vasculature. Very low pressure. More pressure in pushes more force out. Did you notice the change in wording? We don't want to filter fluid in the pulmonary vasculature. That would be called pulmonary edema, and it would compromise the diffusion barrier. But fluid came out of the glomerular capillary because there were fenestrations, because filtration was the point. In the pulmonary vasculature, where there is a tight endothelial junction, the **pushing force from within the vessels opens those vessels**. An increase in pulmonary blood flow, an increase in the blood delivered by the right ventricle, will cause arterioles, capillaries, and venules to inflate, staying open.

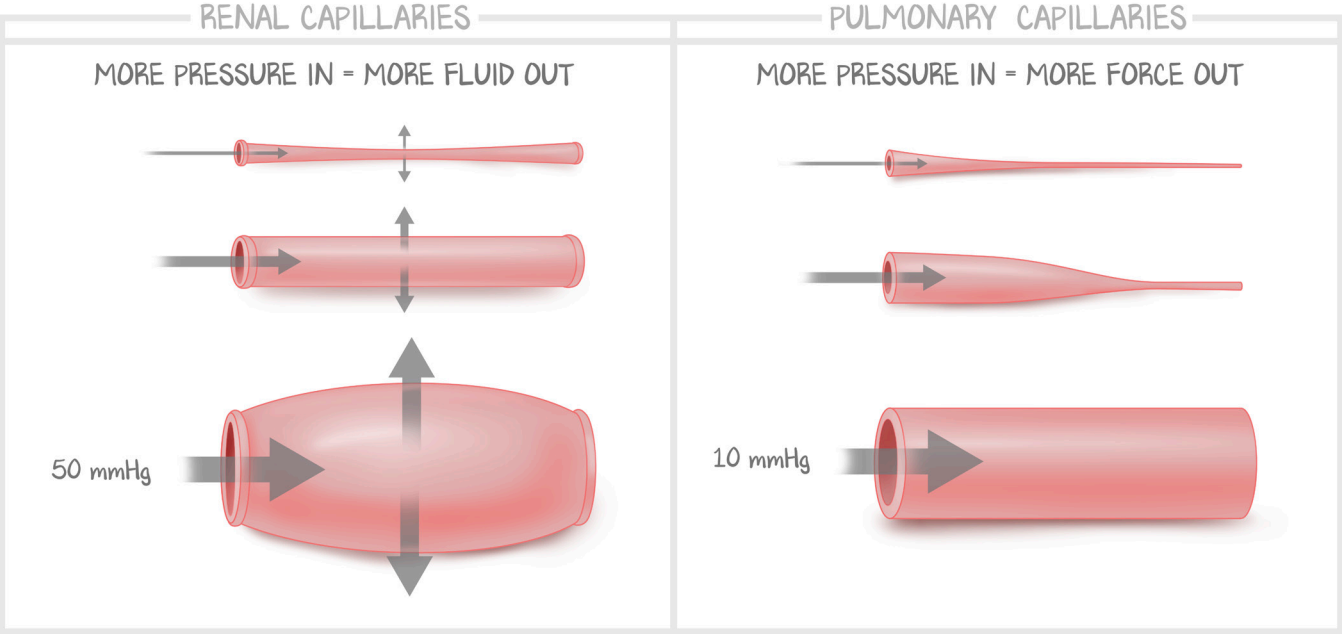


**Figure 1.4: Hydrostatic Forces of the Capillary**
In the glomerular capillary, more renal blood flow causes more hydrostatic pressure and more fluid to be filtered into the tubules. In the already low-pressure system of the pulmonary circulation, an increase in blood flow also increases hydrostatic pressure, but just enough to keep the vessel open and blood running through it.

## Pulmonary Vascular Resistance

Pulmonary vascular resistance (PVR) is a value we calculate when we are assessing someone who might have pulmonary hypertension. In the next lesson, we'll talk about PVR: the value, how we calculate it, how a physician actually gets the values used to calculate it, and what it means. For now, PVR is simply the resistance of the pulmonary vasculature. High resistance, low flow. Low resistance, high flow. Resistance is determined by the caliber of the lumen of all the vessels put together. **All the blood vessels put together** is the key here. The resistance through the pulmonary vasculature is the sum of the resistances.

You got the whole "expiration dilates arterioles and compresses the capillaries" bit. The reverse is true of inspiration. On their own, they make sense. But because both vessels are everywhere in the lung, the consequence is that there is an additive effect. On maximal expiration, there is maximum compression and maximum resistance on the arterioles. On maximal inspiration, there is maximum resistance on the capillaries. This means that at maximum expiration (also called the **residual volume**), the resistance through the circuit is **really high**. It also means that at maximum inspiration (also called the **total lung capacity**), the resistance through the circuit is **also really high**.

The sweet spot, where the resistance to pulmonary blood flow is the lowest, where both the arterioles and capillaries are the least compressed, is at the functional residual capacity, the volume of air at the

bottom of the tidal volume, the end of normal quiet respiration. And because the tidal volume isn't that big, the resistance doesn't change all that much. The **lowest pulmonary vascular resistance is found at normal tidal volume**.

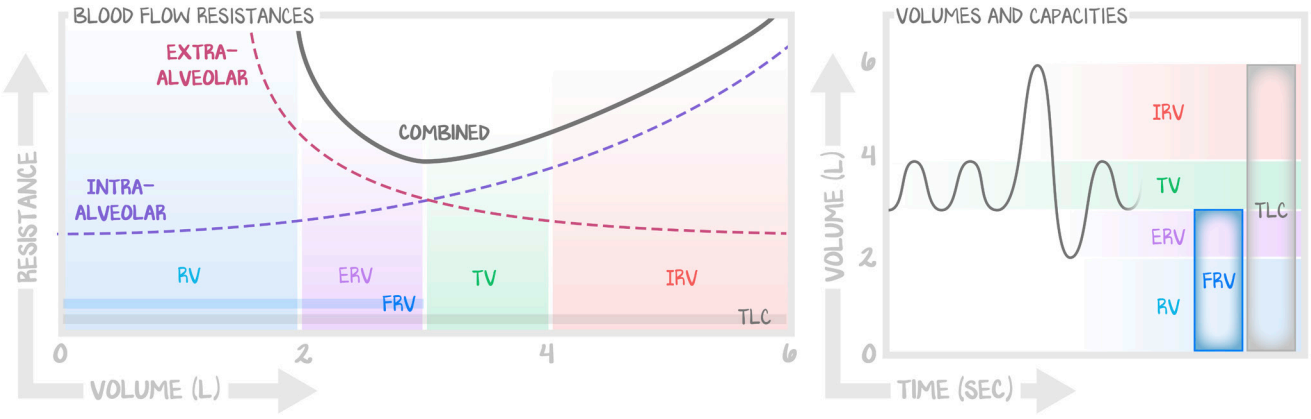


**Figure 1.5: Combined Resistances**
Resistance to blood flow is lowest at the volumes of quiet respiration. Isn't that convenient?

The pulmonary vascular resistance is maximized for perfusion at the lung volumes that a human hangs out at all the time. Pulmonary vascular resistance is supposed to be 24/12. Pulmonary hypertension is what happens when the overall pressure—the overall pulmonary vascular resistance—increases.

## Gravity's Effect on the Pulmonary Vasculature and the Lung Zones

There are two ways to look at this gravity phenomenon—with lots of complicated math (which we're not going to do), or with a simplified version (which we are going to do). We're not going to use transmural pressures and gradients from barometric pressure. Let's use what you know already to make this really easy. We'll use it in greater complexity in the next lesson. But let's make sure to understand the concept now.

**Gravity pulls down.** The diaphragm pulls down. Pulling on alveoli opens them. **Alveoli** have weight and can **push down** on those below. Pushing on alveoli compresses them. All alveoli get the same gravitational pull, the same amount of force that favors the alveoli's opening. The alveoli on top of the lung feel that opening force and no weight from any alveoli above them. The alveoli at the bottom of the lung feel that gravitational opening force and all of the weight from all the alveoli above them. Which, then, is going to open more? The ones on the top. Which open less? The ones on the bottom. The ones on the bottom are relatively compressed compared to the ones on the top.

Now, did you see how we haven't said anything about zones, apex, or base? There are not three lung zones. There is a continuum of pressure differences such that the alveoli transition gradually, from most open on top to least open on the bottom. When **upright**, that change occurs **from apex to base**. But in any position, **the part of the lung farthest from the ground** is the most open.

Capillaries get compressed (resistance increases) when alveoli open, and capillaries get opened (resistance decreases) when alveoli close. Because the alveoli at the top are the most open, the **capillaries at the top have the highest resistance**. Because the alveoli at the bottom are the least open, the **capillaries at the bottom have the lowest resistance**. That means that blood flow is lowest at the top and highest at the bottom.

The lung zones are artificial constructs, theoretical representations of relative pressures that enable the discussion of recruitment and dilation that follows. Some arterioles are collapsed. Some are open but not conducting because the venule on the other side is collapsed. Some are open and conducting because neither is collapsed. Capillary hydrostatic pressure pushes out. Alveoli push in.

In **Zone 1**, both the arteriolar side of the capillary and the venule side of the capillary are collapsed. The reason is that the alveoli have opened the most and have compressed the vessels the most. The alveoli pushing in win. In zone 1, the resistance is maximal and the flow through these vessels is the least. This is the zone of real clinical significance. If there is a compromise in blood pressure, as occurs in shock or hemorrhage, the systemic pressure falls. So, too can the pulmonary pressure. If there isn't enough blood pressure, the alveoli perfused by these capillaries will not be perfused and cannot participate in gas exchange.

In **Zone 2**, the arteriolar side of the capillary has enough hydrostatic force pushing out that it opens up the capillary. But just like in all capillaries, that hydrostatic force is used across the length of the capillary. In zone 2, there isn't enough hydrostatic force to resist the alveoli's inward push for the entire length of the capillary. On the venule side of the capillary, the pressure from the alveoli collapses the venule. The capillaries are open but are not conducting.

In **Zone 3**, the arteriolar side of the capillary has enough hydrostatic force pushing out that it opens up the capillary and has enough hydrostatic force to keep the capillary open across its full length. These vessels are open and are conducting. Perfusion is maximal in zone 3 and maximized at the bottom of the lung.

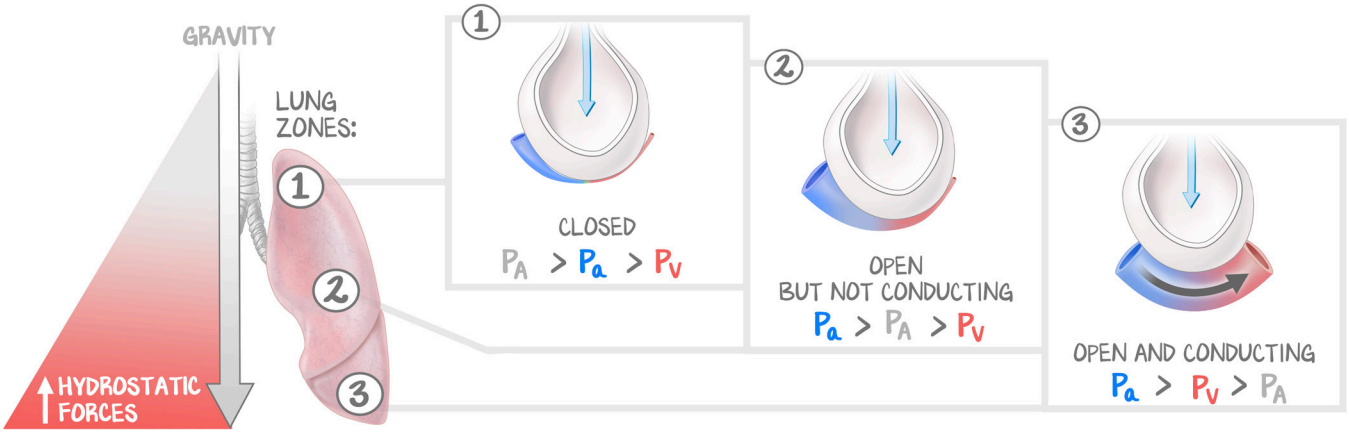


**Figure 1.6: Lung Zones and Alveoli States**
Our illustration shows both the representation found in many texts, and what that means—closed arterioles, open and nonconducting arterioles, and open and conducting arterioles.

## Exercise—Recruitment and Dilation

The systemic circulation is a high-pressure system. That pressure keeps the blood vessels open. All vessels are full all the time. The pulmonary circulation is a low-pressure system. Not all the blood vessels are fully open all the time. Not even the ones that are healthy vessels connected to healthy alveoli. This means that at rest, in a healthy person, adequate gas exchange is occurring without using the full capacity of the pulmonary circulation. Which means there is a **reserve**.

Under "resting" conditions (i.e., at relatively low values of pulmonary arterial pressure), some pulmonary capillaries are open and conducting blood. A familiar example is a type of garden hose used to irrigate a flower bed; this hose is closed at its distal end but perforated with dozens of tiny holes. If the water pressure is low, only some of the holes conduct water. They are all open, but water only comes out of a few. Other holes/vessels are outright closed. We saw this in the last section on lung zones.

**During exercise**, the metabolic demands of the tissues go up. More $CO_2$ is produced and more $O_2$ is needed. In response, the **cardiac output increases**. The heart beats harder and faster, increasing the amount of blood circulating through the vasculature. And because the right heart delivers the same cardiac output to the left heart (via the pulmonary circulation) as the left heart delivers to the peripheral tissues, the cardiac output coming from the right ventricle into the pulmonary circulation also increases. Increased renal blood flow increases GFR by adding an outward force. Increased pulmonary blood flow also adds an increased outward force. Those vessels that were closed are opened, a process called **recruitment**. Those that were open and nonconducting are now more **dilated** and become conducting. More of the lung becomes like zone 3, with more vessels filling and flowing with blood. More arterioles fill with more blood, meaning there are more capillaries carrying deoxygenated blood across more alveoli.

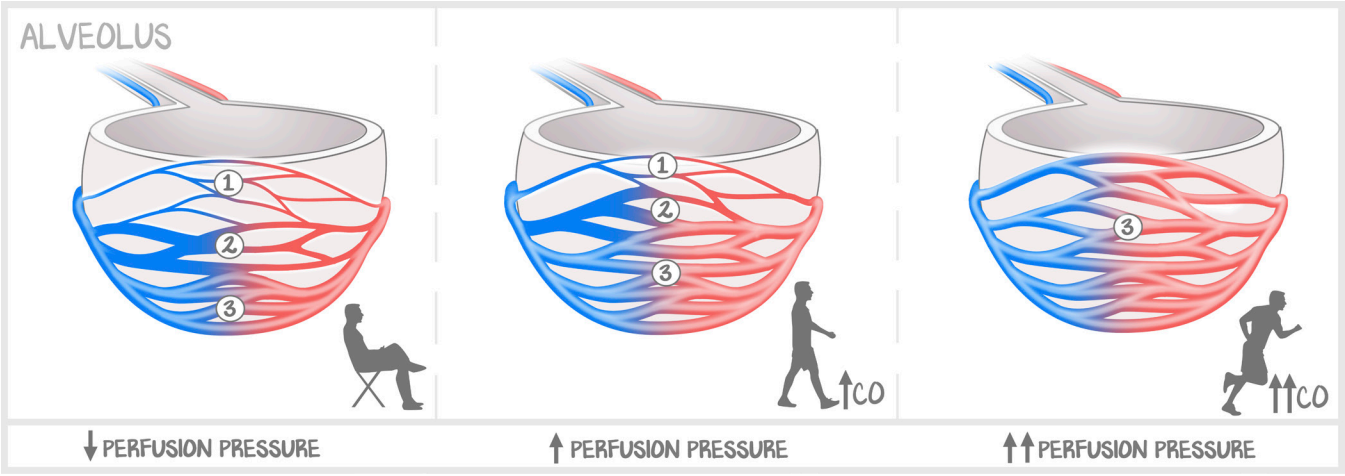


**Figure 1.7: Recruitment and Distension**
Some vessels begin collapsed, others are open and nonconducting. As perfusion pressure increases, the nonconducting vessels become conducting, and the collapsed vessels become open but nonconducting. As perfusion pressure increases even more, all the vessels become conducting. All blood vessels become more like zone 3 as more and more perfusion opens more and more capillaries.

**More blood vessels mean more surface area.** And because the equation that matters at the alveoli is the diffusion equation, increasing the surface area improves gas exchange. More oxygen on, more $CO_2$ off. Exercise is an easy example. But it isn't just exercise that increases cardiac output. Anything that causes the heart rate to go up or the contractions to beat stronger (other than compensation for loss of volume) will increase the cardiac output and increase the pulmonary perfusion pressure, and the lung will undergo recruitment and vessel dilation. The collapsed and nonconducting vessels aren't pathologic states. They are functional reserves of gas exchange the body doesn't need at rest. Chances are, if the cardiac output increases, the metabolic demands are also increased, and so, getting more oxygen into the blood and getting more carbon dioxide out, seems like a good idea.

## Response to Hypoxia

In the periphery, there are mechanisms for signaling to hemoglobin that the tissues need oxygen and separate mechanisms for signaling the arteriole to change its resistance. **Low oxygen levels** in the tissues signal hemoglobin to unload oxygen and also signal the arteriole to **dilate**. **High $CO_2$** levels in the tissues signal hemoglobin to unload oxygen and also signal the arteriole to **dilate**. Hypoxemia, in the tissues, leads to the dilation of the arterioles, reduced resistance, and increased blood flow.

In the **lung**, **hypoxia** "of the tissues" induces **vasoconstriction**. This mechanism is local to the alveoli, free of influence from systemic mediators of vascular tone, and not controlled by innervation. Medical science has not elucidated how this happens. But it is obvious why it happens. Low oxygen levels "in the tissues" at the lung means there is a low oxygen level in the alveolus, that this particular alveolus is not being aerated. If there is a low level of oxygen in this alveolus, it won't do a very good job of oxygenating the blood. So, this alveolus tells the arterioles of its capillaries to vasoconstrict. Vasoconstriction reduces the diameter of the vessel, increasing the resistance, thereby decreasing the flow. Blood follows the path of least resistance, and so is likely to go somewhere else. The goal is to send it to a better-oxygenated alveolus, one that isn't hypoxic. We'll see how this can be problematic in Pulmonary: Circulation #3: *Pulmonary Hypertension*.

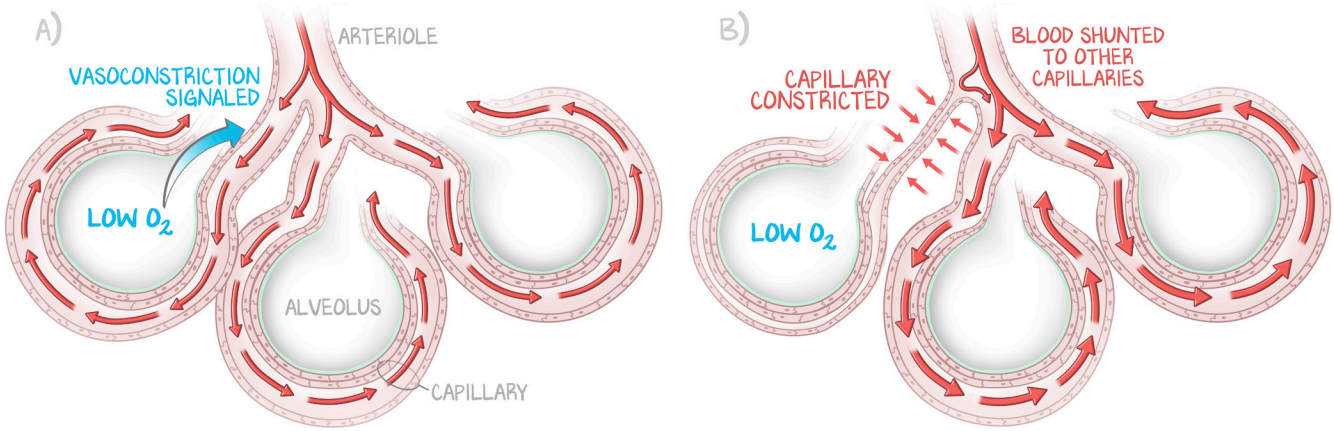


**Figure 1.8: Hypoxemia Drives Vasoconstriction to Shunt Blood**
When an alveolus is poorly aerated, oxygen goes down and carbon dioxide goes up. It makes sense to send that blood somewhere else. To do that, vasoconstriction increases resistance, decreasing the flow and shunting that blood flow to another alveolus. This alveolus doesn't know what's up with other alveoli, but it does know it lacks oxygen, so the blood should try somewhere else.

**Low oxygen levels in an alveolus induce vasoconstriction of the arterioles that feed its capillaries.** This is especially relevant for the next lesson on pulmonary hypertension, as most of the mechanisms involve hypoxia-induced vasoconstriction.

## Extra Physiology Stuff That Everyone Teaches

V/Q mismatch and dead space are relevant to pulmonary physiology. It's just that, because we've explained things without math or many equations, these two otherwise intuitive subjects become unnecessary. We talk about them because we're obligated to. Really, they are just an aside.

## V/Q Mismatch

The purpose of the lungs' capillaries is to perform **gas exchange**. Venous blood returning from the systemic vasculature is carrying deoxygenated blood. The partial pressure of oxygen in the veins is around 40 mmHg (the $P_VO_2$ is 40). The partial pressure of carbon dioxide in the veins is normally around 46 mmHg. In the inspired air, there are approximately 150 mmHg of $O_2$ and 0 mmHg of $CO_2$. In the alveoli, gas exchange occurs. Alveolus and capillary equilibrate the partial pressures of $CO_2$ and O2.

The partial pressure of carbon dioxide in the capillary leaving the alveolus is 40 mmHg. The normal partial pressure of carbon dioxide in the arteries, the **$PaCO_2$, is 40 mmHg.** That makes sense.

The blood leaving the capillary of an alveolus has a partial pressure of oxygen of 100 mmHg. A normal partial pressure of oxygen in the arteries, the **$PaO_2$, is 80 mmHg**, not 100 mmHg. This discrepancy can be explained by the zones of the lung and the ventilation/perfusion mismatch. At the top of the lung, lots of oxygen, not a lot of blood flow. At the bottom of the lung, less oxygen, more blood flow. It isn't that the $PAO_2$ is 150 mmHg everywhere, it's just that that's about how much there is all over the lung. As ventilation and perfusion become more aligned, as the blood flow and ventilation max out, the $PaO_2$ can get a little higher than 80 mmHg. There is no utility in knowing this and no way of using it to diagnose or treat disease.

There is always a V/Q mismatch. Always. All the time. Just as the perfusion had a progression from top to bottom, so too does ventilation. The alveoli on the top of the lung are already open, have elastic recoil pulling to collapse them, and are, therefore, harder to ventilate. The alveoli at the bottom of the lung are the least open, and so have the most ability to ventilate. That's convenient: the alveoli with the most perfusion also get the most ventilation and those with the least perfusion get the least ventilation. At rest.

The thing is, the degree of the change in perfusion from the top to the bottom of the lung is different than the degree of the change in ventilation from the top to the bottom of the lung. Perfusion changes more. V is ventilation; Q is perfusion. At the bottom of the lung, there is more perfusion than ventilation. At the top of the lung, there is less of both, but there is more ventilation than perfusion.

You already knew that. It is *because* the alveoli are inflated that the capillaries are compressed. It isn't a pathologic state. It just is. The body doesn't need a V/Q ratio of 1, to have ventilation and perfusion matched. In fact, it can't. Because of gravity. Because of the zones of the lung.

When the cardiac output increases, when recruitment and dilation happen, when the capillaries open more equally across the zones of the lung, they push outward on the alveoli on the top just as much as the alveoli on the bottom. As cardiac output increases, the V/Q ratio across the lung approaches 1. It's just a mathematical representation of all the blood vessels being used equally.

## Dead Space

Dead space refers to volume and location—it is the combination of words that means the volume of air inhaled with each breath that does not participate in gas exchange, and also where that air is.

The **anatomic dead space** refers to the conducting airways and nasopharynx. Since these structures need to be filled with air before the alveoli can get any, each breath passes air through the anatomic dead space. Although normal ventilation is approximately 500 cc, the anatomic dead space is about 150 cc.

**Physiologic dead space** is the anatomic dead space within alveoli that are aerated but still don't participate in gas exchange. In order to participate in gas exchange, blood has to flow from the arterial side to the venule side of the capillaries. In the last section, we just explained that there is normally physiologic dead space and that it represents a reserve, not a pathologic state.

**Pathologic dead space** is extra dead space, an extra volume of air that does not participate in gas exchange. To create pathologic dead space, we could add additional length to the conducting airways. Ever see someone on a ventilator? Where is the end of the tube, usually? Taped to the side of their face, with a spacer, a long hose, and then connected to the machine that boops and beeps. **Ventilator tubing** increases the dead space. The other way to add pathologic dead space is to **remove capillaries** from participating in gas exchange. That last statement is not intuitive to most learners. We are talking about ventilation. Ventilation is obviously about moving air into and out of alveoli. But the definition of dead space is the volume of air that does not participate in gas exchange. The air is present in the alveoli. If the air is there and yet it doesn't participate in gas exchange, the problem must be with the other player in gas exchange, the blood. In this kind of physiologic dead space, the alveoli are full of air, but the capillaries are collapsed.